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Demystifying wound infection: identification and management

Wound infection delays healing and impairs quality of life. Leanne Cook and Karen Ousey discuss the recognition and treatment of wound infections, which are essential skills for practice nurses.

Wound infection can be costly both in terms of delayed healing and the detrimental effect on the patient’s quality of life. High Quality Care for All (Department of Health (DH), 2008) envisaged putting quality at the heart of everything the NHS does, therefore the ability to recognize and treat wound infections is an essential skill for each and every practitioner dealing with wound management.

Wound infection is a common surgical complication. Surgical site infections are associated with considerable morbidity and it has been reported that over one third of post-operative deaths are related, at least in part, to surgical site infections (Astagneau, 2001).

Surgical site infections account for 15% of all health-care-associated infections (Health Protection Agency (HPA), 2009). They are also associated with considerable morbidity and estimated to at least double the length of hospital stay, thereby increasing the costs of health care (HPA, 2009).

Additional costs attributable to surgical site infections of between £814 and £6626 have been reported depending on the type of surgery and the severity of the infection (National Collaborating Centre for Women’s and Children’s Health (NCC-WCH), 2008).

The main additional costs are related to repeat operation, extra nursing care and interventions, and drug treatment costs. The indirect costs, owing to loss of productivity, patient dissatisfaction and litigation, and reduced quality of life, have been studied less extensively (NCC-WCH, 2008).

The HPA (2009) reported that during 2008, 1191 surgical site infections were detected from 94 750 surgical procedures in 251 hospitals (including NHS and private). About 30% of the surgical site infections required readmission.

Wound infection

Wound infection occurs as a result of a dynamic interaction between the host and the pathogen (White, 2009). All wounds are contaminated with a variety of microorganisms.

Contamination refers to the presence of organisms on the surface of the wound (Stotts, 2004). Often these microbes are harmless and are naturally found on the surface of the skin; these are known as skin flora. Intact skin forms a physical barrier against microbes and many other bacteria but once this defence mechanism is broken, with the creation of a wound, bacteria are provided with a perfect environment to grow and multiply, i.e. a warm, moist surface with plenty of nutrients available.

Bacterial load or burden is an important concept in the understanding of wound infection. Kingsley (2001) described the notion of a continuum in the development of wound infection from sterility to infection. Infection occurs when the sum of the bacterial burden is greater than the host’s immune defences, leading to a systematic immunological reaction.

There are four states in the development of microbial infection (Table 1):

- Contamination
- Colonization
- Critical colonization
- Wound infection.

Bacteria are present in all wounds. Where the number of bacteria in a wound is low (contamination), there is no impairment of wound healing. As the number of bacteria in the wound rises, infection

### Table 1. The four microbial states of the wound infection continuum

<table>
<thead>
<tr>
<th>Term</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Contamination</td>
<td>Bacteria are present on the surface of the wound but do not multiply and do not cause an immune response</td>
</tr>
<tr>
<td>Colonization</td>
<td>The presence of multiplying bacteria in a wound is balanced, or is kept in check by the patient’s immune system. The bacteria do not interfere with wound healing and do not damage wound tissue or trigger an immune response. A normally healing wound is colonized with bacteria</td>
</tr>
<tr>
<td>Critical colonization</td>
<td>The point at which the patient’s immune response can no longer control the colonizing bacteria in the wound, resulting in delayed wound healing</td>
</tr>
<tr>
<td>Infection</td>
<td>The presence of multiplying bacteria which overwhelm the patient’s immune system, disrupting healing and damaging wound tissue and the host’s immune response</td>
</tr>
</tbody>
</table>

becomes more likely. At the state of critical colonization, the wound’s bacterial burden reaches an imbalance, provoking infection if the bacterial burden is not effectively managed (Kingsley, 2001) (Figure 1).

The pathogens most commonly associated with wound infections in the UK are Staphylococcus aureus, Streptococcus species, Pseudomonas aeruginosa and anaerobes (Cooper, 2005).

Infection can occur on acute wounds such as surgical wounds (surgical site infections) and on chronic wounds such as pressure ulcers, diabetic foot ulcer and leg ulcers, which are more likely to be colonized with bacteria due to the nature of the open wound and tissue type (Vazquez-Boland et al, 2006).

Classification of surgical site infections
Surgical site infections develop within 30 days of an operation or within 1 year if an implant was placed within the wound and the infection appears to be related to the surgery.

The ability to classify surgical site infections by severity is important when reporting and auditing surgical site infection rates and also helps with the management of the wound.

The HPA (2008: 18) defines three types of surgical site infection:

- Superficial incisional infection
- Deep incisional infection
- Organ/space infection.

Superficial incisional infection
A superficial incisional infection is one that occurs within 30 days of surgery and involves only the skin or subcutaneous tissue of the incision. Criteria include purulent draining, positive culture from aspirated fluid or wound swab, evidence of pain, tenderness, local swelling and/or heat and redness.

Deep incisional infection
A deep incisional infection is defined as a surgical site infection involving the deep tissues (i.e. fascial and muscle layers) that occurs within 30 days of surgery if no implant is in place, or within 1 year if an implant is in place and the infection appears to be related to the surgical procedure. Diagnostic criteria include purulent draining from the deep incisional space but not from the organ/space, formation of abscess in or around deep incision, positive culture from aspirate or wound swab, deep incision that spontaneously dehisced or one that has been deliberately opened by a surgeon (Figure 2).

Organ/space infection
Organ/space infection is defined as a surgical site infection involving any part of the anatomy other than the incision. It occurs within 30 days of surgery if no implant is in place, or within 1 year if an implant is in place and the infection appears to be related to the surgical procedure. Organ/space infection is associated with the surgical procedure and in addition has one of the following: purulent discharge into a drain placed into an organ/space, positive culture from aspirated fluid or tissue sample, or abscess formation.

The HPA (2009) provides full guidance to surgical site infection and classification.

Diagnosing infection
The diagnosis of infection is primarily a clinical skill based on a complete and accurate assessment of the wound and the patient in combination with laboratory tests.

Most practitioners would feel comfortable identifying frank infection in a wound but find it more difficult to distinguish between those wounds that are colonized and those that are critically colonized. Depending on the host response to the bacteria even relatively low levels of bacterial burden can impair wound healing (Stephen-Hayes and Toner, 2007).

It is essential that the correct diagnosis of wound infection is made to ensure effective management. Several classical signs and symptoms accompany wound infection but not all wounds will exhibit all these signs at any one time (Table 2). Classical signs include pain, redness (erythema), heat, oedema and purulent exudate.

Cutting et al (2005)

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Cutting et al (2005)

Table 2. Signs and symptoms of wound infection

<table>
<thead>
<tr>
<th>Classic signs of infection</th>
<th>Additional signs of infection</th>
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<tbody>
<tr>
<td>Pyrexia</td>
<td>Delayed healing</td>
</tr>
<tr>
<td>Pain</td>
<td>Dark/discoloured granulation tissue</td>
</tr>
<tr>
<td>Oedema</td>
<td>Fragile wound tissue</td>
</tr>
<tr>
<td>Increased exudate</td>
<td>Malodour</td>
</tr>
<tr>
<td>Inflammation</td>
<td>Cellulitis</td>
</tr>
<tr>
<td>Erythema</td>
<td>Pockering at base of wound</td>
</tr>
<tr>
<td></td>
<td>Abscess formation</td>
</tr>
<tr>
<td></td>
<td>Painful/altered sensation around wound bed</td>
</tr>
</tbody>
</table>

From: Cutting et al, 2005.
described additional and potentially more sensitive criteria for identifying wound infection including abscess formation, cellulitis, discharge, delayed healing (compared with the normal rate of healing for the site and condition), discoloration, friable granulation tissue that bleeds easily, unexpected pain and tenderness, pocketing at the base of the wound, bridging of the epithelium or the base of the wound, and tenesmus.

Investigation: swabs
When there are signs of wound infection, a wound swab should be taken to identify the pathogens involved. It is essential that the swab results are interpreted in the light of the clinical signs and symptoms. However, it is important to know that there is little clinical evidence to support the role of wound swabs in identifying wound infection. The use of a wound swab may identify some or all of the bacteria within the wound, but may not always indicate the clinically significant species (Wounds UK, 2010).

Despite the limitations of wound swabs, they will remain part of clinical practice until more advanced techniques are developed and validated (Wounds UK, 2010). The identification of the infecting microbe helps clarify correct management and is essential for highlighting antibiotic sensitivity.

Identifying patients at greater risk of infection
Individuals at greater risk of wound infections include those who are immunologically compromised, neonates and the elderly (White, 2009).

A patient’s individual immune response influences the effect of the bacteria within the wound. The immune response can be affected by many factors including nutritional status, the health of the circulatory system, metabolic disorders such as diabetes, concurrent infections, and medication, e.g. steroid therapy.

Patients who smoke are also at increased risk of developing wound infections (Kean, 2010). Increased susceptibility to wound infection is thought to be due to delayed epithelialization as a result of reduced white cell response and downgraded inflammatory response, both of which lead to a higher bacterial count in the wound bed (Kean, 2010).

Kean (2010) also suggested that wound dehiscence rates may rise in smokers as a result of abnormal fibroblast morphology, cell adhesion and migration, or from a lack of collagen being deposited and remodelled in the wound bed, leading to poor tensile strength.

The healthier the patient, the more likely that a wound will remain harmlessly contaminated or colonized with microorganisms and the less likely infection is to develop (Patel, 2007).

It is important to understand the relationship between a patient’s immune response and the risk of infection in order to accurately assess individual vulnerability to infection, plan measures to reduce the risks of infection (if possible) and provide the patient with the appropriate and accurate information needed to take measures to reduce the risk.

Wound healing in people with conditions such as diabetes is also impaired. Many factors contribute to wound healing deficiencies in people with diabetes, including decreased or impaired growth factor production, delayed angiogenic response and altered macrophage function. As a result, people with diabetic foot ulceration have a high risk of hospitalization, lower limb amputation, and high mortality rates (Falanga, 2005) (Figure 3).

An awareness of local referral pathways is needed to ensure that such patients are seen in a timely manner by a specialist in the management of diabetic foot ulceration. It is important that all practitioners are aware of their limitations and seek advice from specialists in tissue viability when they feel a wound is not progressing as expected and/or where infection in not being controlled.

Management
Correction of the bacterial burden reduces inflammation in the wound bed and therefore promotes healing.

The use of a topical antimicrobial dressing can help control bacterial burden. Antimicrobial dressings are designed to reduce the number of pathogens on the wound bed to a level that no longer impairs wound healing.

There are many antimicrobial dressings on the market. These include silver, honey, polyhexamethylene biguanide (PHMB), inadine and chlorhexidine (Table 3).

<table>
<thead>
<tr>
<th>Table 3. Wound dressing options</th>
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</thead>
<tbody>
<tr>
<td><strong>Antimicrobial</strong></td>
</tr>
<tr>
<td>Silver</td>
</tr>
<tr>
<td>Honey</td>
</tr>
<tr>
<td>Polyhexamethylene biguanide (PHMB)</td>
</tr>
<tr>
<td>Inadine</td>
</tr>
<tr>
<td>Chlorhexidine</td>
</tr>
</tbody>
</table>

From: Joint Formulary Committee, 2011.
Part 2: Infection

All these dressing have different physical properties and currently there is no clear evidence or guidance to indicate which product is better suited to which type of wound or tissue type. However, Wounds UK (2010) has produced a best practice statement on the use of topical antiseptic/antimicrobial agents designed to provide guidance for health practitioners on when to start—and equally important—when to stop using topical antimicrobial agents.

Wounds UK (2010) recommends that in locally infected wounds where there are no signs of the infection spreading, topical antiseptic or antimicrobial agents should be used. If the signs of infection subside and the patient shows no signs of systemic infection, the antiseptic/antimicrobial agent should be discontinued.

In a health service that has to account for the cost-effectiveness of wound dressings, it is important to observe that, although antimicrobial dressings may appear to be expensive, they may be more cost-effective in the long term. In an audit of 133 562 individuals, McDermott-Scales et al (2009) found that 66.7% of patients who received wound-related antibiotics had more than one course. Thus the use of antimicrobial dressings may be both clinically and economically effective when used appropriately.

Systemic antibiotics may not be the most appropriate way to reduce bacterial burden in wounds, particularly with an increase in bacteria resistant to antibiotics. Indeed, Howell-Jones et al (2006) reported that general practices prescribed more antibiotics for patients with chronic wounds than for those who did not have a chronic wound.

Consideration should be given to other methods of reducing the bacterial burden, including tissue debridement, wound cleansing, and increased frequency of dressing changes. This should be done in combination with methods to enhance patients’ resistance to infection and reducing risk factors by ensuring that underlying vascular disease has been addressed, nutritional intake is optimized, and oedema is controlled, encouraging smoking cessation, and supporting optimum control of blood sugar levels in people with diabetes.

Conclusions

The diagnosis of infection and critical colonization in wounds remains a process of recognition and interpretation of clinical signs and symptoms. Practitioners need to have an understanding of this process and the treatments available, as wound infection continues to be a challenge and has a significant impact in terms of quality of life and NHS financial burden. Early recognition of infection, along with prompt and effective treatment improves the quality of patients’ care. It also reduces cost; in the current economic climate, providing cost-effective care is the responsibility of every NHS practitioner.

Conflict of interest: None declared

References